AMENDMENTS TO THE CLAIMS

Claims 1-29 (canceled)

30. (Currently amended) A method of inducing remodeling of cardiac muscle cell sarcomeric and cytoskeleton structures or cell-cell adhesions, which method comprises contacting cardiac muscle cells with a neuregulin protein, ~~comprising an EGF-like domain, or a neuregulin protein comprising an EGF-like domain and an additional amino acid sequence from a wild-type neuregulin protein~~ consisting of an amino acid sequence set forth in SEQ ID NO:2, in an amount sufficient to activate the MAP kinase pathway in said cardiac muscle cells and induce remodeling of said cardiac muscle cell sarcomeric and cytoskeleton structures or cell-cell adhesions.

31-36. (canceled)

37. (previously presented) The method of claim 30, wherein the neuregulin protein is used in an amount that is at least 10^{-8} M.

38. (previously presented) The method of claim 30, wherein the cardiomyocyte or the cardiac muscle cells exist in a mammal.

39. (previously presented) The method of claim 38, wherein the mammal is a human.

40. (previously presented) The method of claim 39, wherein the human has or is suspected of having a heart failure.

41. (previously presented) The method of claim 40, wherein the heart failure is a disease state selected from the group consisting of congestive heart failure, myocardial infarction, tachyarrythmia, familial hypertrophic cardiomyopathy, ischaemic heart disease, idiopathic dilated cardiomyopathy and myocarditis.

42. (previously presented) The method of claim 40, wherein the heart failure is in the form of ischaemic, congenital, rheumatic, or idiopathic.

43. (previously presented) The method of claim 40, wherein the heart failure results from disassociation of cardiac muscle cell-cell adhesion and/or the disarray of sarcomeric structures in the mammal.

44. (previously presented) The method of claim 38, wherein the neuregulin protein is administered with a pharmaceutically acceptable carrier or excipient.

45. (previously presented) The method of claim 30, wherein the contact of the cardiac muscle cells with the neuregulin protein decreases DNA synthesis in the cardiac muscle cells.

46. (previously presented) The method of claim 30, wherein the contact of the cardiac muscle cells with the neuregulin protein results in sustained activation of the MAP kinase pathway in the cardiac muscle cells.

47. (previously presented) The method of claim 38, wherein the neuregulin protein is administered orally, using a sustained-release system or via injection or infusion.

48. (previously presented) The method of claim 47, wherein the injection or infusion is selected from the group consisting of intravenous, intraperitoneal, intracerebral, intramuscular, intraocular, intraarterial and intralesional injection or infusion.

49. (previously presented) The method of claim 30, which further comprises contacting the cardiac muscle cells with an effective amount of an agent which causes cardiac hypertrophy or congestive heart failure.

50. (previously presented) The method of claim 49, wherein the agent which causes cardiac hypertrophy or congestive heart failure is fludrocortisone acetate or herceptin.

51. (previously presented) The method of claim 30, which further comprises contacting the cardiac muscle cells with an effective amount of an agent that acts to suppress a hypertrophy induction pathway different from the pathway suppressed by the neuregulin.

52. (previously presented) The method of claim 51, wherein the agent that acts to suppress a hypertrophy induction pathway different from the pathway suppressed by the neuregulin is selected from the group consisting of a cardiotrophic inhibitor, an angiotensin-converting enzyme (ACE) inhibitor, a human growth hormone, an IGF-I, an anti-hypertrophic, myocardiotrophic factor, an anti-arrhythmic factor and an inotropic factor.

53. (previously presented) The method of claim 30, which further comprises contacting the cardiac muscle cells with an effective amount of an angiotensin-converting enzyme (ACE) inhibitor.

54. (previously presented) The method of claim 53, wherein the ACE inhibitor is selected from the group consisting of quinapril, ramipril, captopril, benazepril, fosinopril, lisinopril, enalapril and lisinopril.

55. (previously presented) The method of claim 30, which further comprises contacting the cardiac muscle cells with an effective amount of an agent for treating hypertension.

56. (previously presented) The method of claim 55, wherein the agent for treating hypertension is selected from the group consisting of an antibody to the endothelin receptor, a β-adrenoreceptor antagonist, an α_1-noreceptor antagonist, an anti-oxidant, a β-blocker and a growth hormone.

57. (Currently amended) A method for treating or delaying disassociation of cardiac muscle cell-cell adhesion and/or the disarray of sarcomeric structures in a mammal, which method comprises administering to a mammal, to which such treatment or delay is needed or desirable, a neuregulin protein, ~~comprising an EGF-like domain, or a neuregulin protein comprising an EGF-~~

~~like domain and an additional amino acid sequence from a wild-type neuregulin protein~~ consisting of an amino acid sequence set forth in SEQ ID NO:2, in an amount sufficient to activate the MAP kinase pathway in said mammal, whereby said disassociation of cardiac muscle cell-cell adhesion and/or the disarray of sarcomeric structures is treated or delayed in said mammal.

58. (canceled)

59. (new) The method of claim 57, wherein the mammal is a human.

60. (new) The method of claim 59, wherein the human has or is suspected of having a heart failure.

61. (new) The method of claim 60, wherein the heart failure is a disease state selected from the group consisting of congestive heart failure, myocardial infarction, tachyarrythmia, familial hypertrophic cardiomyopathy, ischaemic heart disease, idiopathic dilated cardiomyopathy and myocarditis.

62. (new) The method of claim 60, wherein the heart failure is in the form of ischaemic, congenital, rheumatic, or idiopathic.

63. (new) The method of claim 57, wherein administration of the neuregulin protein decreases DNA synthesis in the cardiac muscle cells of the mammal.

64. (new) The method of claim 57, wherein administration of the neuregulin protein results in sustained activation of the MAP kinase pathway in the cardiac muscle cells of the mammal.